A CASE OF DYSPHAGIA AND DYSPHASIA RESULTING FROM A LESION IN THE INTERNAL CAPSULE.[1]

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This patient, a married man, fifty-six years of age, was seen in consultation with Dr. W. H. Warder on September 25th, 1896, when the following history was obtained: There is nothing of importance in his family or personal history until five years ago when he suffered from repeated attacks of gout or rheumatism, which more particularly affected the left foot. Soon afterwards he was considerably shocked by the death of his father, and later was much worried by business troubles. Upon April 8th, 1894, at one A. M., he awoke with intense pain in the back of the head, and shortly afterwards, while evacuating the bowels, he fainted. This intense pain continued several weeks despite treatment. Seven months later he had a similar, though milder attack. Two years and four months ago he was under the care of Dr. Joseph Leidy, Jr., and at this time, while evacuating the bowels, his wife found him mumbling in an unintelligible manner, which made it impossible for her to understand what he desired to express. He was able to walk, and there was no paralysis of the arms or legs. There was interference with the movement of the tongue and the left side of the face with considerable dysphagia, which seemed to be dependent upon the condition of the pharyngeal muscles, especially the constrictors. There was

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no true aphasia. He made a complete recovery in four weeks. About this time he was subject to attacks of polyarticular rheumatism, and from time to time suffered with indigestion. About fourteen months ago he developed dyspnea with marked irregularity of respiratory movements, which later became pronounced Cheyne-Stokes breathing. This was accompanied by edema, more especially of the lower extremities, and at that time his physician recognized the existence of cardiovascular and renal disease.

At the time of my first examination Cheyne-Stokes breathing was well marked. There was moderate edema of the lower extremities. The heart showed considerable hypertrophy of the left ventricle, but the sounds were clear and all arteries accessible to touch showed well-marked thickening from fibrosis and atheroma.

A summary of twelve urinary analyses shows that the quantity varied between thirty-three and forty-three ounces, that the color was lighter than normal, that the albumin varied from one to two per cent., that there was no sugar, that there was usually present slight indicanuria, that the specific gravity varied between 1008 and 1011, that the reaction was decidedly acid, that the urea was 8/10ths of one per cent., and that at first no, and later a few, narrow hyaline tube casts were present. Shortly before death the hyaline tube casts increased in number, and a few granular casts were also found.

The lungs showed a moderate grade of emphysema. The digestion was feeble, and he was limited to the simplest food. Any slight indiscretion in diet would produce marked gastric disturbance and even nausea. The liver was slightly enlarged. The speech was at times hurried and enunciation somewhat mumbling, but a part of this defect in speech was due to rapid and irregular breathing.

The eye-grounds were repeatedly and carefully examined by Dr. Thomas H. Fenton, who found that the nerve heads were slightly elevated and pale, that the veins were dilated, and that the arteries were contracted. There

were no disturbances of the visual fields, and acuity of vision was normal.

Cheyne-Stokes breathing continued more or less well marked until five or six weeks before death, when it suddenly disappeared. He died suddenly on January 26th, 1897, from heart failure.

The autopsy was performed Jan. 28th, 1897, with the following results: The heart and blood vessels attached weighed nineteen ounces, and the heart was very much enlarged in all its diameters. The left ventricle, which was

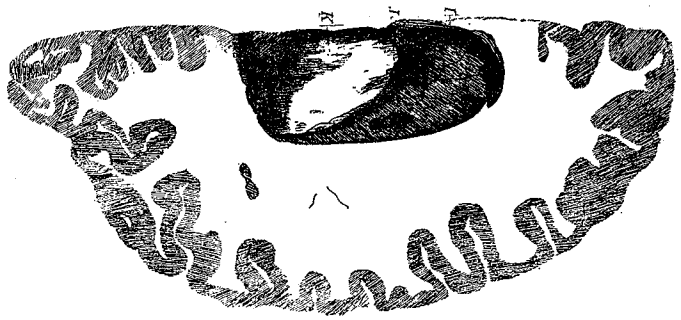

FIG. I.

I, Caudate Nucleus; *J*, Small Lesion in the Caudate Nucleus; *K*, Thalamus.

dilated and hypertrophied, constituted the major part of the cardiac enlargement. The mitral valves and orifice were normal. All visible arteries showed marked atheromatous changes. The inferior vena cava was of large size and partially empty. The pericardium was normal.

The aortic leaflets were competent, moderately thickened, but otherwise normal. The tricuspid orifice was dilated, but the valves were normal as were also the pulmonary valves. At the extreme lowermost portion of the left ventricular cavity, at a point corresponding to the apex, were a moderate dilatation and thinning of the walls, and the ventricle contained an organized thrombus of about

the size of a large marble. Superimposed was a small and more recent thrombus. Some of the chorda tendineæ attached to the mitral valve were attenuated and elongated. The base of the anterior leaflet of the mitral valve, near the base of the aortic leaflet, showed several patches of atheroma which were yellow in color. The aorta was thickened and dilated, and pouched posteriorly and to the right. There were numerous atheromatous patches beneath the intima. The spleen was at least three times its normal size, and was adherent posteriorly. The structure showed no macroscopical change. The left kidney

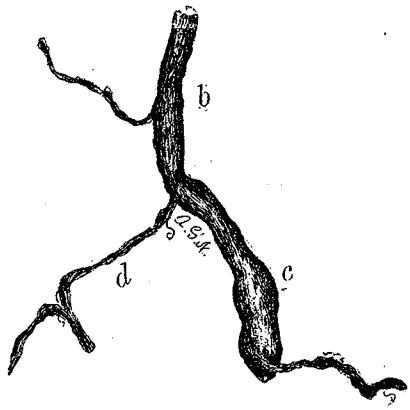

FIG. II.

b, Basilar Artery; *c*, Right Vertebral Artery; *d*, Left Vertebral Artery.

was smaller than normal, and the capsule was adherent, but with care could be detached from the cortex without tearing the renal structure. The entire surface presented a markedly granular appearance, and the superior portion was occupied by a large cyst the size of a walnut. Over the surface were two or three smaller cysts containing a clear liquid. Upon section the cortex was narrowed and showed the presence of a large amount of fibroid tissue. The pyramids were smaller than normal, and showed marked fibroid changes. The ureters were normal. The

right kidney was in a similar condition. The condition of the kidneys was the result of a long-continued, chronic, interstitial and parenchymatous nephritis. The gastro-intestinal tract showed no abnormality.

The liver was enlarged and projected four fingers' breadth below the edge of the ribs. On section it presented the characteristic nutmeg appearance, and showed evidence of considerable fibrosis and moderate fatty degeneration.

The calvarium was unusually thick anteriorly. The sinuses were empty, and there was no evidence of inflammatory change in the meninges. There was a moderate amount of effusion beneath the meninges and within the ventricles and the spinal canal. The convolutions of the brain were well developed and appeared normal. Upon section the white substance appeared normal, and the red spots corresponding to sections of dilated vessels were numerous and of considerable size.

About the middle of the caudate nucleus and cutting or pressing upon the fibres passing to the knee of the right internal capsule was a lesion of about $\frac{1}{4}$ of an inch in diameter and $\frac{1}{8}$ of an inch in depth, which showed the remains of a hemorrhage which probably occurred two years and four months previously. This lesion was plainly apparent on opening the right ventricles, and is well shown in Fig. I.

The blood vessels all presented marked evidence of atheroma. The right vertebral and basilar arteries, especially the former, were aneurysmally dilated and pouched, and their calibre was fully as large as an ordinary carotid (Fig. II.). In places the wall was extremely thin and almost transparent. This aneurysm was close to the medulla oblongata and was quite capable of causing a certain degree of pressure upon the neighboring tissues. The left vertebral was unusually small. The posterior meningeal arteries showed atheroma, but no marked change in the lumen.

This case is interesting as showing (1) that a small

superficial lesion situated close to the knee of the right internal capsule is capable of producing dysphagia and dysphasia; (2) the appearances presented by a small hemorrhage two years and four months after its occurrence; the symptoms produced and their variations during this period; (3) that the wide-spread and advancing arterio-capillary fibrosis and atheroma, probably rheumatic in origin, produced the customary cardiovascular and renal changes, and also the aneurysm of the right vertebral and basilar arteries; (4) the long continuance of the Cheyne-Stokes breathing on and off for four or five months, which was probably in part due to cardiac failure and slight uremia.

The aneurysm of the vertebral and basilar arteries, pressing upon the medulla, raises the question as to whether this also may not have been a contributory factor in the production of the symptoms.

The situation of the small aneurysm was such that it could readily have compressed the right glossopharyngeal and hypoglossal nerves, so that the difficulty in swallowing could in part be explained in this manner.

PARALYSIS OF RIGHT THIRD NERVE FROM HEMORRHAGIC NEURITIS WITH EXTRAVASATION OF BLOOD OVER THE OPPOSITE FRONTAL LOBE.

In the Edinburgh Medical Journal, for May, 1897, Gibson and Turner report the case of a girl eleven months old, admitted to the hospital, for diarrhœa and vomiting, with wasting, who had right-sided ptosis, with dilation and immobility of the pupil, and inability to turn the right eye inward. Her temperature ran an irregular course, her pulse and respiration were rapid, and she died after five days, having presented no more definite symptoms. The autopsy showed a hemorrhagic neuritis of the right third nerve, the left being nearly normal. On the left side of the brain there was a large hemorrhage, occupying the area over the middle third of the Sylvian fissure, with a prolongation upwards and forwards involving the bases of the frontal gyri, especially that of the second. The centrum ovale and gray matter about the Sylvian aqueduct showed nothing abnormal. The authors regard the nerve lesion as entirely responsible for the eye palsy. No mention is made of the condition of other organs. C. L. ALLEN.